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MODULATION OF HEART TISSUE DAMAGE IN CHAGASIC MICE BY INTERLEUKIN-4. Soares, M.B.P.; Lima, R.S.; Silva-Mota, K.N.; Bellintani, M.C.; Pontes de Carvalho, L.C.; Ribeiro dos Santos, R. Centro de Pesquisas Gonçalo Moniz, FIOCRUZ-BA

Objective: To investigate the role of IL-4 in the development of carditis in the experimental Chagas' disease.

Methods: Infection with 100 Colombian strain *T.cruzi* of wild-type and IL-4 -/- BALB/c mice were compared rearding of parasitemia, mortality, histhology and immune response.

Results: Infected IL-4 +/+ BALB/c mice had higher parasitemia and a mortality than IL-4 -/- BALB/c mice in the acute phase. Histopathology of hearts from IL-4 +/+ mice revealed the presence of multifocal inflammatory infiltrate by mononuclear cells and intense tissue parasitism 30 days post-infection (dpi). In contrast, hearts of IL-4 -/- mice had carditis 2-3 fold more intense and lower tissue parasitism than wild-type mice. Interestingly, hearts obtained from IL-4 +/+ mice after 3 months post-infection presented discrete and focal carditis, whereas in IL-4 -/- mice carditis was intense. At 7 months of infection, wild-type mice presented lesions in the heart characteristic of chronic phase. However, IL-4 -/- mice were 2-3 fold more intense myocarditis at this timepoint. Parasitemia and tissue parasitism in the heart sections examined were negative in both groups after 3 months of infection. IL-4 -/- BALB/c mice presented higher levels of IFN-gamma than wild-type mice in response to *T.cruzi* antigen. Anti-*T.cruzi* IgG titers were higher for IgG1 in sera from IL-4 +/+ mice, whereas IL-4 -/- mice produced higher levels of IgG3 anti-*T.cruzi* antibodies.

Conclusion: These results indicate a regulatory role of IL-4 in the development of chagasic cardiomyopathy.